Symposium*

Dehydration-induced drinking: peripheral and central aspects

Introduction

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The physiological mechanism that induces water intake in mammals is incompletely understood. At least six distinct stimuli have been used experimentally for this purpose: *1*) hypertonic saline; *2*) β-adrenergic agonists, especially isoproterenol; *3*) the octapeptide, angiotensin II; *4*) polyethylene glycol; *5*) parasympathomimetic agents; and *6*) dehydration. The diversity of these stimuli and the differences in their responsiveness from different routes of administration suggest the difficulty in ascribing the induction of water intake to a single physiological mechanism. Indeed, with such a basic function as drinking, there is likely more than one underlying mechanism.

Oatley has proposed a model of drinking in which two separate pathways, osmotic and angiotensin II, are identified where the stimuli for water intake associated with each pathway appear to be additive and not interactive (Fig. 1). Stimuli that affect extracellular fluid (ECF) volume, such as hemorrhage, dehydration, and administration of polyethylene glycol or hypertonic saline, can initiate drinking. Stimuli that affect ECF osmolality, such as dehydration and administration of hypertonic saline, also affect the intracellular fluid (ICF) volume. The classical technique to induce drinking is water deprivation with its consequent dehydration, where the ECF volume decreases and plasma osmolality increases. Increases in the osmolality and sodium concentration of ECF are adequate stimuli to induce drinking by way of stimulation of osmoreceptors or sodium receptors. Discussion of the location and function of these receptors will be presented in the symposium.

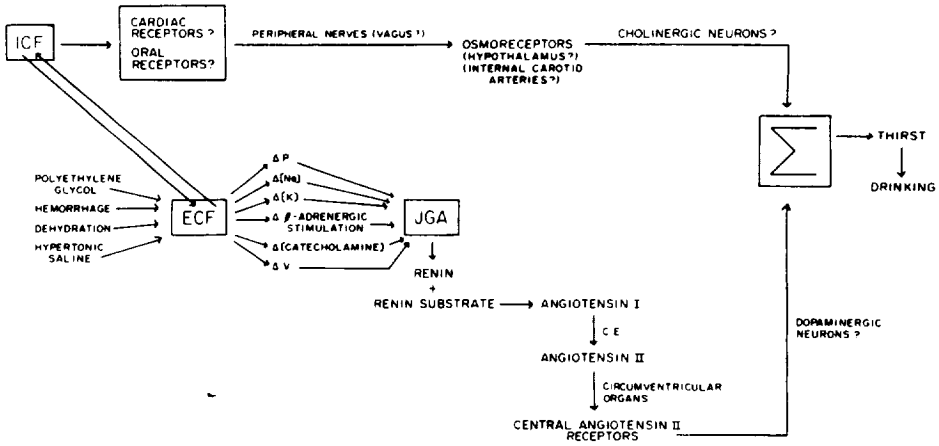

Figure 1. A model illustrating two major pathways—osmoreceptors (above) and angiotensin (below), and associated factors for induction of fluid intake. Synthesized from Oatley, K. Simulation and theory of thirst. Epstein, A. N.; Kissileff, H. R.; Stellar, E., eds. *The neuropsychology of thirst: new findings and advances in concepts*. Washington, DC: Winston & Sons; 1973: 199–223.

Despite the appeal to explain dehydration-induced drinking as a phenomenon mediated by osmoreceptor or sodium receptor, the angiotensin II pathway also plays a role (Fig. 1). Other stimuli that induce drinking, such

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as a decrease in blood pressure (ΔP) and/or volume (ΔV), a decrease in plasma sodium concentration ($\Delta[Na]$), an increase in plasma potassium concentration ($\Delta[K]$), or β-adrenergic stimulation via adrenergic nerves or plasma, initiate release of renin from the kidneys. After the release of renin into the blood, angiotensin II is formed and gains access to receptors in the brain. Dopaminergic neurons are presumed to mediate this response. Both pathways summate to induce drinking (Fig. 1). However, the relative importance of each pathway is unknown. Some parts of this dual pathway hypothesis will be discussed in relation to drinking by humans and rats exposed to stressful environmental conditions. The important problem of thirst satiation and termination of drinking will be addressed in the final paper.

The scientific study of the induction and termination of fluid intake has come of age. We hope this symposium will generate additional interest and stimulate the formation of novel hypotheses that will lead to a clearer understanding of this vitally important aspect of body fluid regulation.